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Investigation of Sperm Count and Motility as a Cause of Male Infertility in District Malakand, Khyber Pakhtunkhwa, Pakistan

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Abstract

Introduction: Infertility is a major global public health concern affecting millions of couples worldwide. Male-related factors contribute to approximately half of all infertility cases, with abnormalities in semen parameters, including sperm concentration, motility, and morphology, representing major contributors to male infertility. District Malakand, like many regions of Pakistan, lacks comprehensive data on patterns of male infertility and associated semen abnormalities. Therefore, this study was designed to investigate sperm-related causes of male infertility among infertile men in District Malakand by evaluating key semen parameters, including sperm count, motility, morphology, and viability. **Methodology:** A total of 19 infertile males from five families were enrolled in the study. Semen samples were collected under standardized laboratory conditions and analyzed in accordance with World Health Organization (WHO) guidelines. The evaluated parameters included semen volume, sperm count, motility, morphology, viability, and the presence of pus cells. **Results:** The results revealed considerable variation in semen quality among the affected individuals. Azoospermia was observed in 29.6% of infertile males, indicating complete absence of sperm in semen. Oligospermia characterized by reduced sperm concentration was identified in 22.2% of individuals. Normospermia with functional impairment was detected in 51.8% of cases, suggesting that normal semen parameters do not always guarantee fertility. Morphological abnormalities were identified in 18.5% of individuals. **Conclusion:** Abnormalities in sperm concentration, motility, morphology, and viability were strongly associated with infertility in the studied population. Comprehensive semen analysis is essential for the accurate assessment and diagnosis of male infertility. Further clinical and molecular investigations are warranted to better understand the underlying factors contributing to male infertility in this population.

Introduction

Infertility, referring as the inability to conceive after 12-14 months of regular unprotected intercourse, is a multifactorial condition with profound medical, psychological, and social consequences (Kamel, 2010). Infertility is classified into primary (no prior pregnancy), secondary (inability after previous pregnancy), and subfertility (delayed conception beyond one year) (Gnoth et al., 2005; Khan et al., 2007). Male factors account for approximately 25% of infertility cases, female factors for 35%, combined factors for 30%, while 15% remain unexplained (Khan et al., 2007). In Pakistan, infertility prevalence is estimated at 21.9%, with primary infertility at 3.5% and secondary infertility at 18.4% (Khan et al., 2007). Semen analysis remains the cornerstone of male fertility assessment, evaluating sperm concentration, motility, morphology, and viability. Oligospermia (sperm count <20 million/mL) and azoospermia (complete absence of spermatozoa) represent major causes of male infertility while asthenospermia involves impaired motility despite normal concentration (Khan et al., 2007).

The etiology extends beyond sperm abnormalities to include structural defects, endocrine dysfunction, genetic disorders, environmental exposures, and infections (Nieschlag et al., 2023). Genetic abnormalities, particularly Y chromosome microdeletions involving AZF regions, are significant causes of spermatogenic failure (Chang et al., 2005; Elliott & Cooke, 1998). Genes such as *CDY* and *TSPY* play critical roles in spermatogenesis (Vogt, 2004). Chromosomal abnormalities contribute to 5% of oligospermia and 10% of azoospermia cases (Ck, 2001). Varicocele, characterized by pampiniform plexus dilatation, impairs testicular physiology and sperm production (Musalam et al., 2010). The hypothalamic pituitary gonadal axis regulates spermatogenesis through LH, FSH, prolactin, and testosterone; disturbances in this axis impair fertility (Babu et al., 2004; Khan et al., 2007). Environmental pollutants, heavy metals, and occupational exposures adversely affect sperm quality (Selevan et al., 2000).

District Malakand, like many regions of Pakistan, lacks comprehensive epidemiological data on male infertility and associated semen abnormalities. Therefore, the present study was designed to investigate sperm-related causes of male infertility among infertile men in District Malakand by evaluating key semen parameters, including sperm count, motility, and morphology. The findings of this study are expected to enhance the understanding of local patterns of male infertility and contribute to the development of evidence-based strategies for its diagnosis, management, and prevention in the region.

Materials and Methods

Study Population

The present study included five families (MIF-01, MIF-02, MIF-03, MIF-04, and MIF-05) from District Malakand, Khyber Pakhtunkhwa, Pakistan. Families were recruited based on the presence of male infertility, with female-related causes of infertility excluded. Demographic and clinical information was collected through structured interviews and review of available medical records.

Ethical Approval

The present study was approved by Institutional Bioethics Committee at Government Postgraduate College Dargai, Malakand (BEC No. 195), and was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki (2013). All study procedures involving human participants were performed in accordance with the approved ethical guidelines and institutional requirements.

Pedigree Construction

Pedigrees spanning three to four generations were constructed according to standardized human pedigree nomenclature (Bennett et al., 2022). In the pedigrees, squares represented males and circles represented females, while filled symbols denoted affected individuals and unfilled symbols indicated unaffected individuals. Non-consanguineous and Consanguineous marriages were represented by single and double lines connecting the respective partners.

Semen Collection and Analysis

Semen samples were collected following 3-4 days of sexual abstinence in sterile, labelled containers. Following collection, semen samples were maintained at 37°C and analyzed within one hour of liquefaction in accordance with WHO guidelines (2010). Macroscopic evaluation included assessment of semen volume, color, viscosity, and pH. Microscopic examination was performed at 200× and 400× magnification to assess sperm aggregation, pus cells, and morphological abnormalities. Sperm concentration was determined using a Neubauer hemocytometer. Sperm motility was classified as progressively motile, sluggishly motile, or non-

motile, based on the assessment of 100 spermatozoa across three microscopic fields for each sample. Sperm morphology was evaluated by examining 100 spermatozoa per specimen according to WHO criteria (2010). The reference values used for semen analysis included a semen volume of ≥2.0 mL, sperm concentration of 20-130 million/mL, progressive motility of ≥45%, dead spermatozoa of ≤5%, and pus cell count of ≤5/HPF.

Results

Family Collection

In the present study, a total of five families comprising 19 infertile male individuals were investigated belonging to District Malakand, Khyber Pakhtunkhwa, Pakistan. The families and individuals were identified through visits to their residences, infertility clinics, local communities, and other relevant sources. Participants were interviewed regarding their history of infertility and inability to conceive. Following confirmation of infertility, semen samples were collected from affected individuals and fertile controls. For each family, detailed pedigrees were constructed based on interviews with affected individuals and senior family members to document the familial distribution and inheritance patterns of infertility.

Clinical Features

Nineteen infertile males from five families (MIF-01-MIF-05) were investigated in the present study. All affected individuals presented with infertility as the primary clinical manifestation, while exhibiting normal height, skin, and sexual maturation.

Family MIF-01

Family MIF-01 comprised 19 members spanning three generations, including two affected males (III-6 and III-8) (Figure 1). Both affected individuals exhibited normal sperm concentrations but showed increased proportions of sluggishly motile and non-viable spermatozoa, consistent with normospermia accompanied by functional sperm impairments. Mild evidence of infection was also observed in both individuals (Table 1).

Family MIF-02

Family MIF-02 comprised 25 members spanning four generations, with six affected individuals (II-6, III-5, III-6, III-8, IV-2, and VI-4) (Figure 1). Semen analysis revealed azoospermia in two affected individuals (III-6 and III-8), whereas individual IV-2 exhibited oligospermia. The remaining affected individuals showed normospermia accompanied by functional sperm impairments (Table 1).

Family MIF-03

Family MIF-03 comprised 22 members spanning four generations, including six affected individuals (II-6, III-2, III-4, III-6, III-8, and IV-1) (Figure 1). Semen analysis revealed a high proportion of sluggishly motile and non-viable spermatozoa in three affected individuals (III-4, III-6, and III-8), indicating impaired sperm motility and viability (Table 1).

Family MIF-04

Family MIF-04 comprised 11 members spanning three generations, with two affected individuals (II-3 and II-5) (Figure 1). Semen analysis revealed oligospermia in the affected individuals (Table 1).

Family MIF-05

MIF-05 comprised 13 members spanning three generations, with three affected individuals (III-1, III-3, and III-5) (Figure 1). Semen analysis revealed markedly

impaired semen quality, with affected individuals presenting with astheno-necrozoospermia, asthenozoospermia, and oligospermia (Table 1). Among the 16 infertile males investigated, azoospermia was identified in 29.6% of individuals, oligospermia in 22.2%, morphological abnormalities in 18.5%, and normospermia with functional impairment in 51.8% of cases.

Family ID	Patient ID	Status	Semen Color	Semen Volume (mL)	Viscosity	Motile Sperm (%)	Sluggish Sperm (%)	Dead Sperm (%)	Sperm Count (mill/mL)	Pus Cells (HPF)	Final Diagnosis
Normal Reference	-	-	Whitish	≥ 2.0	Thin	≥ 45	0–5	0–5	20–130	0-5	Normal
MIF-01	III-6	Affected	White turbid	2.0	Thin	60	20	10	80	1-2	Normospermia with Functional Impairment
	III-8	Affected	Whitish	1.5	Thin	52	13	20	75	10-15	Normospermia with Functional Impairment
	III-5	Normal	White turbid	2.0	Thin	80	10	10	80	1-2	Normal
MIF-02	III-6	Affected	Watery	0.1	Thin	Nil	Nil	Nil	Nil	1-2	Azoospermia
	III-8	Affected	Watery	0.3	Thin	Nil	Nil	100	Nil	1-2	Azoospermia
	IV-2	Affected	Whitish	2.5	Thick	25	15	60	80	4-10	Oligospermia
	IV-5	Normal	White turbid	2.0	Thin	80	10	10	90	1-2	Normal
MIF-03	III-4	Affected	Whitish	1.5	Thin	60	20	20	50	3-4	Increased Sluggish & Dead Sperm
	III-6	Affected	White turbid	2.0	Thick	68	20	12	40	2-3	Increased Sluggish & Dead Sperm

	III-8	Affected	Grey dirty	2.5	Thick	55	30	15	45	1-2	Increased Sluggish & Dead Sperm
	IV-9	Normal	Whitish	2.0	Thin	80	15	5	80	1-2	Normal
MIF-04	II-3	Affected	Creamy	2.8	Thick	10	10	80	65	10-15	Oligospermia
	II-5	Affected	Grey dirty	2.0	Thin	30	15	40	60	15-20	Oligospermia
	II-1	Normal	Whitish	2.5	Thin	80	10	10	90	1-2	Normal
MIF-05	III-1	Affected	Creamy	2.0	Thick	20	10	70	55	1-2	Astheno-necrozoospermia
	III-3	Affected	Grey dirty	2.6	Thick	70	20	10	38	1-2	Increased Sluggish Sperm
	III-5	Affected	Whitish	1.5	Thin	40	10	50	9	2-5	Oligospermia
	III-6	Normal	White	2.5	Thin	70	10	10	70	1-2	Normal

Table 1. Summarizes the semen parameters of normal**, affected and unaffected individuals from families MIF-01 to MIF-05. Affected individuals exhibited diverse semen abnormalities, including azoospermia, oligospermia, astheno-necrozoospermia, increased sluggish and dead sperm, and normospermia with functional impairment. In contrast, unaffected and carrier individuals generally showed normal semen characteristics, supporting the segregation of male infertility phenotypes with the disease status in the studied families.**

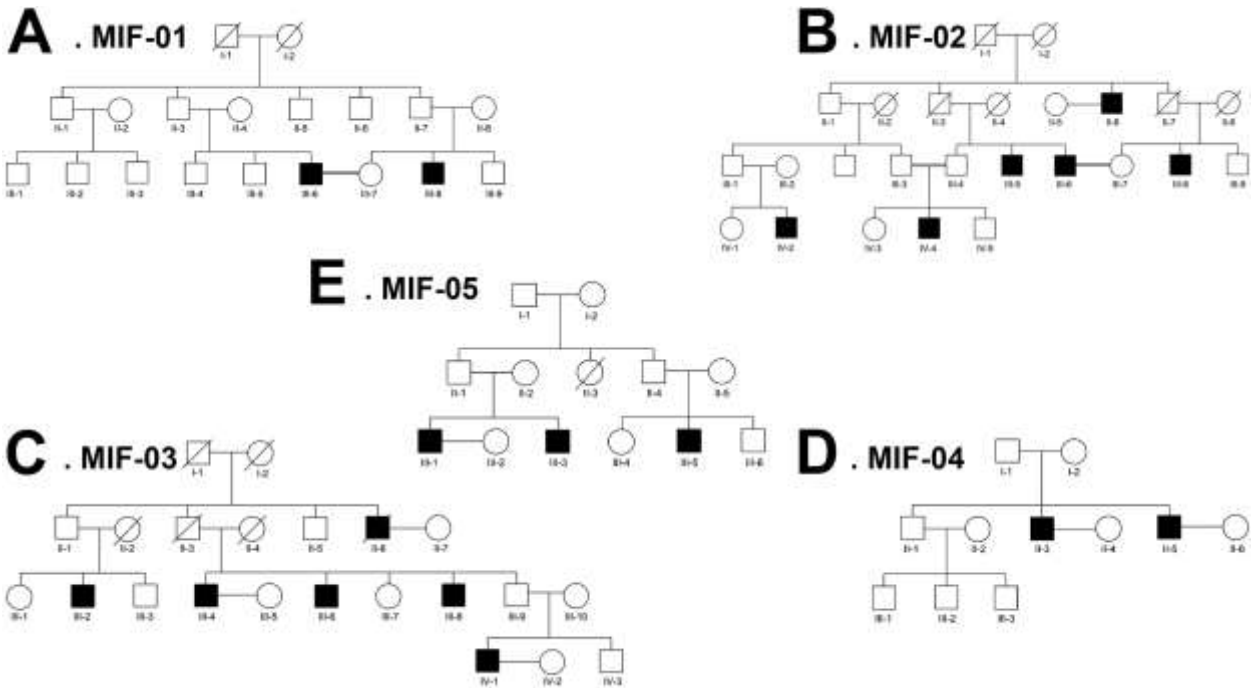


Figure 1. The figure illustrates pedigrees of five consanguineous and non-consanguineous families (MIF-01 to MIF-05) from District Malakand, Khyber Pakhtunkhwa, Pakistan. Square symbols represent males, circles females. Filled symbols indicate affected individuals, open symbols unaffected individuals, and diagonal slashes indicate deceased members. Consanguineous marriages are designated by double lines, non-consanguineous by single lines. Annotations are marked by Arabic numerals, with generations indicated by Roman numerals. **(A)** Family MIF-01 (19 members, 3 generations): two affected males (III-6, III-8) with normospermia with functional impairment. **(B)** Family MIF-02 (25 members across four generations) included six affected males (II-6, III-5, III-6, III-8, IV-2, and IV-4) who exhibited normospermia with sluggish sperm motility. **(C)** Family MIF-03 (22 members across four generations) consisted of six affected individuals (II-6, III-2, III-4, III-6, III-8, and IV-1) who exhibited sluggish sperm motility and non-viable spermatozoa. **(D)** Family MIF-04 (11 members, 3 generations): two affected males (II-3, II-5) with oligospermia. **(E)** Family MIF-05 (13 members, 3 generations): three affected males (III-1, III-3, III-5) with azoospermia, astheno-necrozoospermia, and oligospermia.

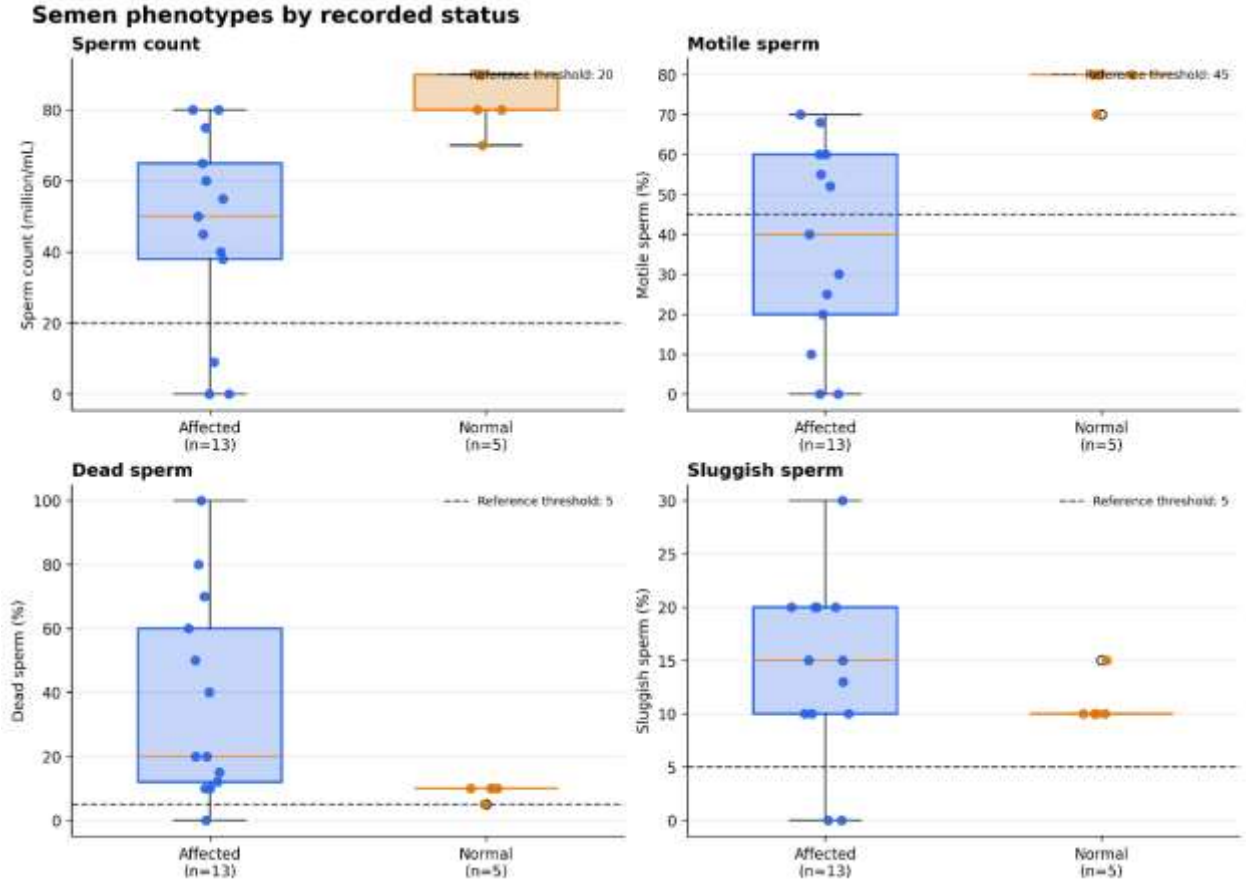


Figure 2. Box plots comparing semen parameters between affected (n = 13) and unaffected (n = 5) individuals. **(A)** Affected individuals exhibited a broader distribution of sperm counts, with several samples falling below the normal reference threshold (20 million/mL), whereas all unaffected individuals remained within the normal range. **(B)** Sperm motility was markedly reduced in affected individuals, with multiple samples below the reference threshold (45%), while unaffected individuals consistently demonstrated normal motility. **(C)** The percentage of dead sperm was substantially higher among affected individuals, frequently exceeding the normal reference limit (5%), whereas unaffected individuals showed consistently low values. **(D)** Similarly, affected individuals displayed elevated percentages of sluggish sperm, with most values exceeding the reference threshold (5%), in contrast to the normal distribution observed in unaffected individuals. Collectively, these findings

demonstrate that affected individuals have significantly impaired semen quality, characterized by reduced sperm count and motility, together with increased sperm death and sluggishness, compared with unaffected controls.

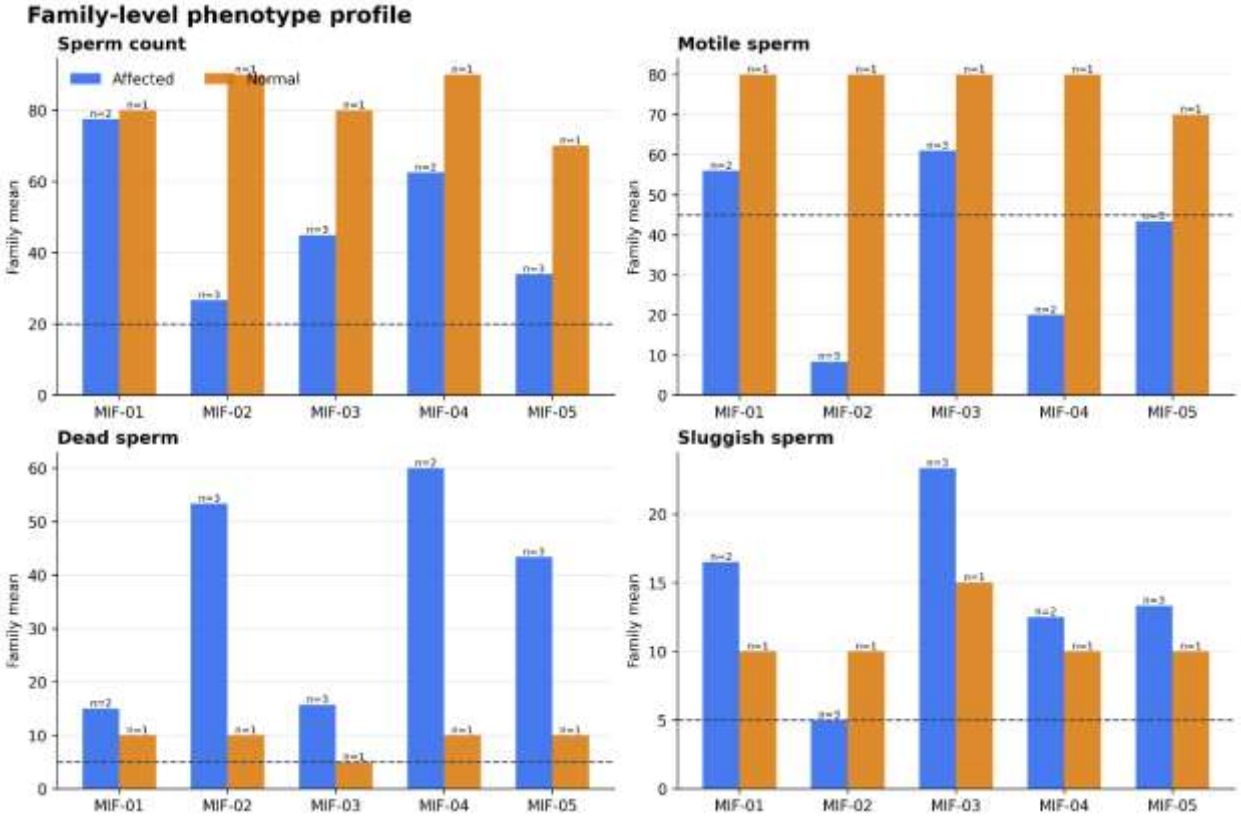


Figure 3. Family-level analysis revealed marked variability in semen phenotypes among affected individuals across families MIF-01 to MIF-05. Affected individuals showed reduced sperm count and motility, accompanied by increased dead and sluggish sperm, whereas unaffected individuals generally exhibited normal semen parameters. The severity of semen abnormalities varied between families, indicating phenotypic heterogeneity in male infertility.

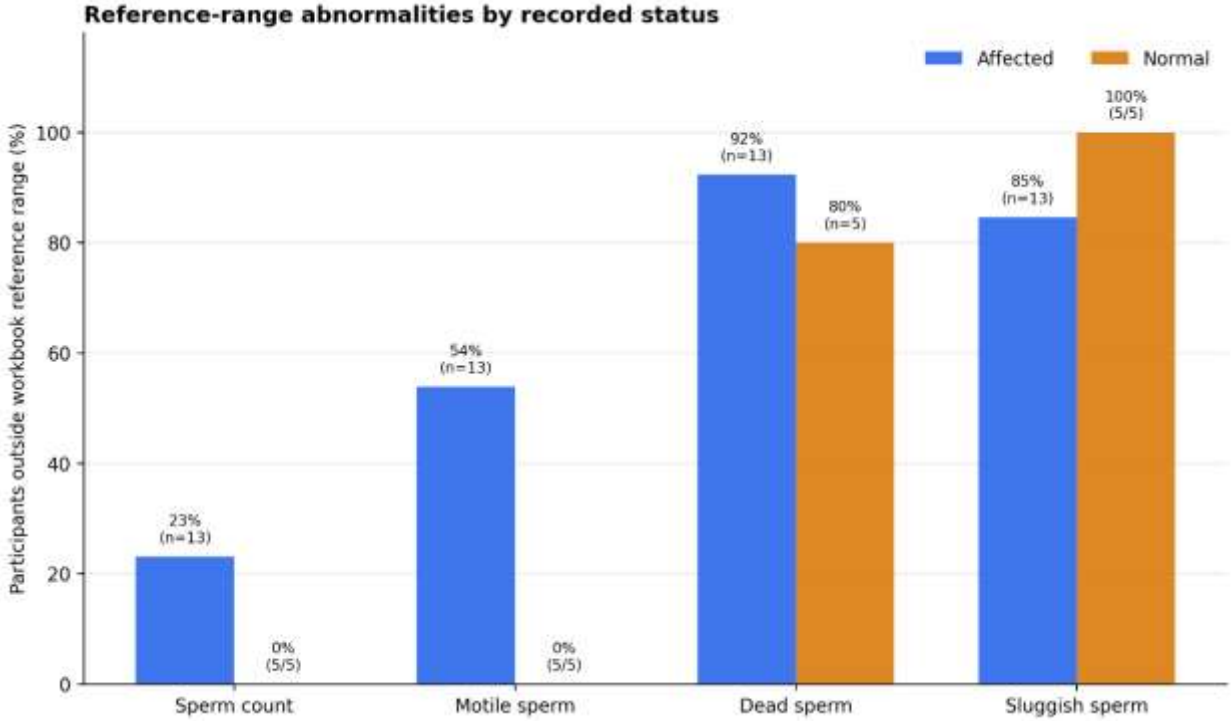


Figure 4. Percentage of participants with semen parameters outside the reference range according to disease status. Compared with unaffected individuals, affected

individuals exhibited a substantially higher frequency of abnormal semen parameters, particularly reduced sperm motility (54% vs. 0%) and decreased sperm count (23% vs. 0%). The highest proportions of abnormalities were observed for dead sperm (92% vs. 80%) and sluggish sperm (85% vs. 100%). Overall, affected individuals demonstrated a greater burden of semen abnormalities, consistent with impaired sperm quality and male infertility.

Discussion

Semen analysis remains fundamental for evaluating male infertility, with sperm concentration serving as a key indicator of reproductive potential. Normal sperm concentration (normozoospermia) exceeds 20 million/mL, while oligospermia and azoospermia represent significant causes of infertility (Khan et al., 2007). Azoospermia is defined as the complete absence of spermatozoa in the ejaculate and represents one of the most severe forms of male infertility (Khan et al., 2007). The findings of the present study demonstrated substantial variation in semen quality among the infertile families investigated in District Malakand. In Family MIF-01, affected individuals exhibited normal sperm concentrations despite infertility, suggesting contributions from impaired motility, abnormal morphology, hormonal disturbances, or genetic defects. Similar observations by Oghagbon (2004) and Abduhadi et al. (2007) reported normospermia in 24.6-25% of infertile males, highlighting the limitations of routine semen analysis (Oghagbon et al., 2004). Family MIF-02 presented more severe abnormalities, with two azoospermia individuals and one oligospermic individual, indicating impaired spermatogenesis as the principal cause. Azoospermia prevalence (29.6%) exceeded the general estimate of 10% (Abdulwaheed et al., 2002) suggesting possible genetic, environmental, or physiological influences.

Family MIF-03 exhibited high proportions of sluggish and non-viable sperm cells despite normal concentrations, indicating qualitative defects in sperm function rather than reduced sperm numbers. Sperm motility is critical for fertilization (Abbas et al., 2020). Family MIF-04 demonstrated oligospermia, consistent with previous reports that 58.8% of infertile males exhibit reduced sperm concentration (Abdulwaheed et al., 2002). Sperm morphology abnormalities (18.5%) further contributed to infertility, as structural defects impair motility and fertilization capacity (Iwamoto et al., 2007). The high prevalence of normospermia with functional impairment (51.8%) exceeds previous reports (24.6-25%) (Jan et al., 2026; Oghagbon et al., 2004), suggesting that factors beyond routine semen parameters, including functional sperm defects, genetic abnormalities, or hormonal imbalances, contribute significantly to infertility. Family MIF-05 affected individuals exhibited normal height, normal hair, and sexual maturity but were characterized by astheno-necrozoospermia, asthenozoospermia, and oligospermia. The presence of infertility exclusively among male family members suggested a possible male-specific inheritance pattern. Hormonal disturbances in the hypothalamic pituitary gonadal axis, particularly reduced testosterone and FSH, impair spermatogenesis (Babu et al., 2004; Khan et al., 2007). However, combined hormonal and seminal analysis provides more reliable assessment (Carosi & Calabro, 1968). Familial clustering and consanguinity suggest possible genetic involvement, consistent with Pakistan's high rates of consanguineous marriages and autosomal recessive disorders. Premarital testing, newborn screening, and parental genetic screening can reduce disease burden (Zaman et al., 2024; Zaman et al., 2023). The heterogeneous spectrum of semen abnormalities observed, including normozoospermia, oligospermia, azoospermia, and impaired sperm motility, underscores the multifactorial nature of male infertility and highlights the importance of comprehensive semen analysis for accurate diagnosis and clinical evaluation.

Conclusion

Male infertility in the investigated population is primarily associated with abnormalities in semen parameters, including sperm concentration, motility, morphology, and viability. Azoospermia (29.6%) and oligospermia (22.2%) indicate impaired spermatogenesis, while morphological abnormalities (18.5%) contribute to reproductive failure. Normospermia with functional impairment (51.8%) suggests that factors beyond routine semen analysis contribute to infertility. Semen analysis remains an essential first-line diagnostic tool, providing valuable information for identifying underlying causes and supporting clinical management. In Pakistan's consanguineous population, premarital testing, newborn screening, and parental genetic screening are recommended to reduce disease burden. Further investigations involving hormonal, genetic, and molecular analyses are warranted to elucidate cases with normal routine parameters but persistent infertility.

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CONFLICT OF INTEREST STATEMENT

The authors declare no relevant financial or non-financial competing interests.

DATA AVAILABILITY STATEMENT

The datasets generated and analyzed during this study are available from the corresponding author upon reasonable request for research purposes only.

ETHICS STATEMENT

This study was approved by the Institutional Bioethics Committee at Government Postgraduate College Dargai, Malakand (BEC No. 195), in accordance with the Declaration of Helsinki (2013). Written informed consent was obtained from all participants and their legal guardians.

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